HÆMATURIA AS A COMPLICATING FACTOR IN APPENDICITIS.

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THE following three eases illustrate the practical difficulties encountered as a result of the development of hæmaturia due to appendicitis.

CASE I.—P. F., 28 years old, a native of Russia, gave a negative family and past history. Present history dates back three months, during the course of which time he had two acute attacks of abdominal pain. These attacks had been diagnosed as appendicitis by a New York surgeon who had also advised operation. A week before admission to the Jewish Hospital of St. Louis he had a similar typical attack of acute appendicitis. When the patient entered the hospital all acute symptoms had subsided, and the physical examination was negative except for a slight residual tenderness over MeBurney's point. The urine at this time was free from all pathological elements. Preparation for appendicectomy was ordered, but about eighteen hours before the time set for operation the patient suffered excrueiating pain in the right lumbar region. His temperature rose a degree and a half, but his pulse-rate remained normal. There was only minimal pain over the appendix, but slight pressure in the lumbar region caused great pain. This lumbar pain remained localized, and did not radiate along the right wreter. An examination of the urine, made at this time, gave the following findings: sp. gr. 1015, color, dark brown, turbid; reaction, acid; albumen, trace; sugar, none; easts, none; blood, abundant (20 to 100 red cells to a field). On the basis that the attack might be due to renal colic rather than to appendicitis, we deferred operation. An X-ray nieture was taken but it did not show the presence of a stone. A eystoscopic examination was negative. No tubercle bacilli were found in the urine. This attack lasted two days, and after its subsidence the patient was observed for three weeks. During this period there were two attacks exactly similar to the first one. Exploratory laparotomy was now decided upon, with the idea of examining the appendix first, and if that organ were found to be normal to expose the right kidney. The operation disclosed a very acutely inflamed appendix. The patient was traced for six months after the appendicectomy, and during this time he was hard at work, manifesting no evidence of renal or intraperitoneal disease.

CASE II.—A. S., 28 years old, Russian, peddler, family and past history negative. Three days before admission to the hospital he had an acute attack of pain limited to the right iliae fossa and right loin, accompanied by fever (101°) and tenderness over the whole right side of the abdomen. There had been no chill, no voniting, and no other symptoms referable to the gastrointestinal tract. The disease had been diagnosed by Dr. Friedman as appendicitis. When I saw the patient his temperature was 100.6°, his pulse 99, and there was slight tenderness over McBurney's point. The abdominal walls were lax. In the right loin, tenderness was exquisitely marked, and at this site there was distinct bulging, over which deep fluctuation could be made out. At the time of this examination the urine was bright red in color. due to the admixture of a very large quantity of blood. An aspirating needle inserted into the bulging mass in the loin withdrew extremely foul-smelling pns. The result of the aspiration, the severe hæmaturia, the laxness of the abdominal walls, the minimal amount of abdominal tenderness, and the absence of marked gastro-intestinal symptoms, led me to make the diagnosis of perinephritic abseess due to primary renal disease. The patient was too septie at this time to attempt to determine the exact nature of the kidney lesion. The perinephritic abscess was opened and drained, and the patient returned to bed. He never rallied from the operation, dying eighteen hours later. A postmortem examination disclosed a general diffuse purulent peritonitis, due to a totally gangrenous perforated appendix. The cæeum lay directly anterior to the right kidney, and the appendix was retrocæcal, lying upon the kidney. The entire perinephritie tissue, including the kidney eapsule, was gangrenous, and the kidney was so intensely eongested that it was a deep

blue-black in color. There were numerous infarctions of the kidney cortex.

CASE III.—I. T., 34 years old, seamstress by occupation. Family history negative. Ten years ago she had a profuse pulmonary hemorrhage and was told by her physician that she had tuberculosis. After a prolonged stay in a Northern resort she was pronounced cured. Present history dated back four months, the chief complaints being frequent painful urination, and continuous backache, with intercurrent acute attacks corresponding in every detail with the symptoms of renal colic. The patient stated that in one of these attacks three months ago she urinated pure blood, but that she never noticed blood in her urine before or after this. The day after her visit to the office she had an acute attack in which she experienced pain in the right loin, radiating down the right ureter. The pain was so severe that she fainted. Physical examination disclosed slight dulness over the apex of the right lung anteriorly, tenderness over the right kidney and along the course of the right ureter, and the presence of a faint trace of albumin and a few red blood-cells in the urine. There was an afternoon temperature of 99.5°. Tubercle bacilli were never discovered in the urine. Ureter catheterization was done by Dr. Johnson, with the following result: The bladder mucosa was normal, as was also the ureteral openings. A ureter catheter readily passed up the left ureter to the kidney. The right ureter was blocked at a point about two inches from the bladder wall. Even a stylet-armed catheter could not be forced by the obstruction.

These findings led to the thought that a ureter stone was causing the obstruction and all the other symptoms already detailed. Five X-ray plates made at three different sittings by Dr. Carman showed in each instance a clear-cut shadow in the course of the intrapelvic portion of the right ureter. Pain was persistent, excruciating, and incessant after the catheterization, and this symptom confirmed us in our belief that a stone had been dislodged from a fairly comfortable resting place.

At operation, the ureter was exposed by the iliac extraperitoneal route, from the kidney to the bladder, but no stone was found in it. At the site where the X-ray showed a shadow, the ureter was kinked as if pulled upward and inward. At the site of kinking there seemed to be a hard nodule resting on the anterior surface of the ureter, and in order to determine exactly what this nodule was the peritoneum was opened. Through this opening we made out that an inflamed appendix containing a stony hard concretion was adherent to the anterior surface of the nreter. At the site of adhesion the ureter was pulled upward and kinked. Appendicectomy was done, the peritoneum sutured, and the wound in the soft parts closed around a drain. An X-ray picture was taken of the appendix immediately, and this picture gave a distinct shadow of the concretion. The appendix was then opened and found to contain a few drops of pus in its dilated tip, back of which there was a dense feeal concretion that had formed about a small seed with a hard chitinous capsule. (The seed was somewhat larger than the seed of a tomato.) The patient reported six months later that she was perfectly well.

Here then, are three eases, all of them encountered within a short period of time, and all of them characterized by the facts, first, that they were wrongly diagnosed by the operating surgeon, secondly, that pain radiating from the kidney region, and blood in the urine were prominent symptoms, and thirdly, that the lesion was in the appendix. By a strange coincidence this set of three eases establishes a basis for a rational elassification of instances of hæmaturia complicating appendicitis. The first ease was one in which no direct relationship could be established between the lesion in the appendix and the hæmaturia. Dieulafoy 3 asserts that there is an intimate relationship between acute appendicitis and nephritis, and he bases his assertion on the elinical observation that acute nephritis so often accompanies acute lesions of the appendix. nephritis, which Diculatoy calls "Nephrite toxique appendieulaire," is supposed to be due to irritation of the kidneys by the toxins resulting from the inflammation of the appendix. Dieulafoy states, furthermore, that the severity of the nephritis is in direct proportion to the acuity of the lesion in the appendix. Whether this last statement be true or not, it certainly is a fact, that acute appendicitis often causes an acute nephritis, and there is no reason for not believing that the inflammation of the kidney may result in the presence of red

blood-eells in the urine. Dieulafoy, in his paper, makes no mention of hæmaturia, but Hildebrand ⁴ in a paper confirming Dieulafoy's observations, records a case of acute appendicitis complicated by a well-marked hæmaturia. In this case, the hæmaturia disappeared after the acute inflammation of the appendix subsided, but reappeared with a second attack of appendicitis, finally disappearing for good, after the appendix was removed. Accepting, then, the views of Dieulafoy and Hildebrand, we may assume fairly that in our first case the hæmaturia was due to a toxic nephritis, secondary to appendicitis.

In our second case, the bleeding was due to a direct involvement of the kidney, as a result of the proximity of an acutely inflamed and gangrenous appendix, which had infected the perinephric fatty and cellular tissue.

In our third ease we know that the ureter was kinked by an adherent appendix, but we cannot state positively just what caused the presence of blood in the urine. Possibly the kinking of the ureter caused a venous obstruction, and a consequent slight outpouring of blood from the ureteral mucous membrane. I can find in the literature only two other cases of appendicitis that caused marked urinary symptoms due to adhesion between the ureter and the appendix. These cases are reported by Lancien, but he makes no mention of hæmaturia as a symptom.

Bearing our three eases in mind, we see how readily they lend themselves to the following elassification: (1) Cases of hæmaturia due to the actions of toxins upon the kidneys; (2) eases of hæmaturia due to direct involvement of the kidney; and (3) eases of hæmaturia due to direct involvement of the ureter. If, in addition to the three cases reported in this paper, we examine the recorded eases in literature, we find one other source of hæmaturia complicating appendicitis. Cases are recorded in which the urinary bladder was perforated by an appendicular absecss, one of the symptoms of the perforation being blood in the urine. (Odde and Silhol,6 Lancien,6 Brun.7)

A complete classification of the subject therefore would have to be framed as follows:

Hæmaturia complicating appendicitis may be due to:

- 1. General systemic invasion resulting from acute appendicitis, and affecting the kidney indirectly,—so-called toxic nephritis.
- 2. Involvement directly of one or more of the organs of the urinary tract.
 - a. Kidney, as in case 2 of this paper.
 - b. Ureter, as in case 3 of this paper.
- c. Bladder, as in the cases recorded by Odde and Silhol, Lancien, and Brun.

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The recorded cases of blood in the urine of patients suffering with appendicitis are very scanty. There are no papers in English, French or German, that take up the subject by title; but the following papers all have a direct bearing on the subject.

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Note.—Since the completion of this paper, an article has been written by Dr. Gray L. Hunner (Jour. Am. Med. Asso., Apr. 25, 1908) emphasizing the importance of hæmaturia as a complicating factor in appendicitis.